TOTTERING MICE: THE KEY TO UNLOCKING THE ROLE OF

ALPHA-1A SUBUNIT CALCIUM CHANNELS ON SPATIAL

LEARNING AND MEMORY

A Senior Honors Thesis

by

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UNIVERSITY UNDERGRADUATE
RESEARCH FELLOWS

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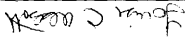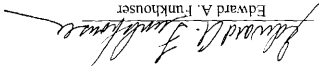
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ABSTRACT

Tottering Mice: The Key to Unlocking the Role of Alpha-1A Subunit Calcium Channels on Spatial Learning and Memory. (April 2001)

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Tottering mice have a mutation in the calcium channel protein, alpha-1A, which decreases calcium entry into neurons through P- and Q-type high-voltage activated calcium ion channels. Because calcium is an important signaling molecule for learning and memory, these mice are appropriate models to study disorders concerning learning and memory. Homozygous tottering (tg/tg) mice exhibit three specific neurological disorders: absence epilepsy, ataxia and paroxysmal dyskinesia. Calcium ions mainly enter neurons through calcium channels that are multisubunit complexes composed of a pore-forming alpha-1 protein subunit and several regulatory subunits. Channels containing alpha-1A subunits (P- and Q-type calcium channels) are highly expressed in the cerebellum, hippocampus, olfactory bulb, cerebral cortex, and thalamus. Since the alpha-1A subunit is found in the hippocampus, and the hippocampus is critical in spatial learning, we postulated that there is a decrease in spatial learning and memory in tottering mice because of the defects in the alpha-1A subunit that they express. However, no investigation of spatial learning has been done on tottering mice.

One problem that needed to be considered is that recurring seizures can interfere with learning and daily tasks. Mice in general swim very well. Swimming seems to eliminate the occurrence of absence seizures and the paroxysmal dyskinesia in tottering mice while the mice are swimming. Therefore, using a swimming maze emphasized

potential learning deficits and not physical impairments of tottering mice. We compared male, wildtype (+/+; control) and homozygous tottering mice (tg/tg) using the Morris water maze as a spatial learning and memory test. After training, we counted the number of direct swims (DS) toward the region where the platform was located (NE quadrant) in one category, and noted random swims (RS) as well. The results showed 62.5% +/+ and 30.77% tg/tg mice fit the DS/NE category. With this evidence, the low percentage of tg/tg mice in the DS/NE category supports our hypothesis that these calcium channel-deficient mice have reduced spatial learning and memory.

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INTRODUCTION

Mice that have a mutation in the alpha-1A subunit protein found in P- and Q-type calcium channels are appropriate models for study of human diseases concerning learning and memory. The generation of mutant mice to serve as animal models of human disease is a powerful tool in understanding the manifestation and mechanisms of human disorders (Crawley, 1999). Tottering mice have a spontaneous recessive mutation that alters the alpha-1A calcium channel subunit and impairs P/Q-type calcium currents in neurons (Steinlein and Noebels, 2000). These mutant mice generally have a normal lifespan when given proper care (Jun et. al., 1999). Previous work from our laboratory has shown that significant morphologic and biochemical abnormalities exist in the tottering mouse cerebellum (Isaacs and Abbott, 1992; 1995; Heckroth and Abbott, 1994; Rhyu et al., 1999). Homozygous tottering (tg/tg) mice exhibit three specific neurological disorders: 1) absence epilepsy (Noebels and Sidman, 1979); 2) ataxia (Green and Sidman, 1962); and 3) a paroxysmal dyskinesia that bears some common attributes of some forms of human myoclonus (Levitt, 1988; Rhyu et al., 1999).

Calcium ions play a major role in many different physiological functions including: synaptic transmitter release and membrane excitability (Hille, 1992) and neurite outgrowth and plasticity (D'Angelo et al., 1994). Neuronal systems for maintaining Ca^{2+} homeostasis are critical for neuronal development and function (Mattson et al., 1991). The main route of extracellular Ca^{2+} entry into neurons is through voltage-gated calcium channels that are multisubunit complexes composed of a pore-forming/voltage-sensing alpha-1 subunit and several regulatory subunits, including alpha 2-delta, beta and gamma (Hofmann et al., 1994). The alpha-1A subunit is particularly associated with P/Q-type calcium channels (Davies and Morris, 1997). Of the six types of alpha-1 subunits, the alpha-1A subunit is the most highly expressed in the brain (Jun, et. al., 1999). Yet, it is not expressed uniformly in all neurons of the brain. The highest

Figure 1. Hippocampus

Mouse brain coronal section. C=cortex. H=hippocampus. Scale bar approximately 2mm.

expression is found in the cerebellum, hippocampus, olfactory bulb, cerebral cortex, and thalamus. High expression of the alpha-1A subunit in the cerebellum is likely to be associated with the occurrence of the ataxia and dyskinesia observed in tottering mice. High expression of the altered alpha-1A subunit in the tottering mouse cerebral cortex and thalamus can be associated with epilepsy. Dimond (1980) reveals that subcortical structures (cingulum, fornix, thalamus, globus pallidus, and hippocampus) seem to play a crucial role in short-term memory. He noted from Drachman and Arbit's study (1966) that patients with bilateral hippocampal lesions had impaired storage abilities (Dimond, 1980). Functionally, Dimond (1980) presents that the hippocampus commits information to memory as well as recalls stored information from memory (Figure 1 shows a section of mouse hippocampus). To emphasize the major underlying point, Crawley (1999) highlights that spatial learning tasks, such as the Morris water maze, require a functional hippocampus. However, no investigation of spatial learning has been done using

tottering mice. We propose to look for any correlation between the expression of abnormal alpha-1A calcium channel subunits in the tottering mouse hippocampus and altered spatial learning and memory.

Hypothesis

Because the alpha-1A protein subunit is highly expressed in neurons of the hippocampus, and the hippocampus is critical in spatial learning, we can postulate that there is a decrease in spatial learning and memory in tottering mice.

Background

Since voltage-gated calcium channels are important for synaptic function, it is possible that the tottering mutation could interfere with synaptic transmission in the hippocampus and, therefore, not allow learning and/or memory to take place. The mice with the alpha-1A calcium channel protein mutation (tottering mice) will be used to explore the acquisition of learning by investigating their behaviors using a Morris water maze. Since the Morris water maze has been standardized in assessing spatial learning and memory, it is an appropriate apparatus to study such behavior in mice (Crawley, 1999). One problem that needed to be taken into account is that recurring absence seizures can interfere with learning and daily tasks. The cerebellum is a highly structured region of the brain and the cerebellar cortex consists of three distinct layers: the outer molecular layer, the Purkinje cell layer, and the granule cell layer (Figure 2). Both granule cells and Purkinje cells highly express the alpha-1A calcium channel subunit as seen in figure 3, in which a section of mouse cerebellum has been immunohistochemically stained using an antibody to the alpha-1A protein. Abnormalities in the function of Purkinje cells and granule cells of the cerebellum due to the mutation in the alpha-1A subunit are thought to be responsible for the cerebellar deficits seen in tottering mice. These deficits include the ataxia as well as the paroxysmal dyskinesia. When an animal exhibits sensory or motor deficits, choice of tasks is limited by the physical inability of the animal (Crawley, 1999). Swimming, instead of walking, seems to eliminate the absence seizures and the paroxysmal

Figure 2. Cerebellum-Low Magnification

Low magnitude sagittal section through a tottering mouse cerebellum. A=anterior cerebellum. P=posterior cerebellum. M=molecular layer. G=granule cell layer. W=white matter. Scale bar approximately 0.5 mm.

Figure 3. Cerebellum-High Magnification

High power magnification of cerebellum. M=molecular layer. Arrow points to Purkinje cell dentrites. P=Purkinje cell body. G=granule cell layer. Scale bar approximately 100 microns.

dyskinesia while the tottering mice are swimming. This allows us to focus on the learning deficits and not so much on the physical impairments of tottering mice.

Furthermore, stress has the ability to affect hippocampal activity (Hölscher, 1999). However, the advantage of the Morris water maze is that, even though stress may be a factor, the animal must swim (perform) because it has no other alternative, whereas a stressed animal will usually not perform on a dry land task (Hölscher, 1999). To make sure that tottering mice do not have problems in vision or judging distance cues, this test will allow us to distinguish between mice that have acquired spatial learning from mice that may not be able to attain spatial learning from other impairments (Silva et al., 1992). The tottering C57BL/6J strain of mouse was utilized in this study because this strain

carries the genetic mutation that produces the three specific neurological disorders mentioned previously. These three disorders have many characteristics of human myoclonus. This commercially available strain serves as the standard for numerous behavioral tasks (Crawley, 1999).

METHODS

We compared wildtype (+/+; control) and homozygous tottering mice (tg/tg) for evidence to test the hypothesis. The tg/tg mice are affected at all alpha-1A subunit calcium channels. To simplify our test results, we only used male mice so hormonal factors would not be a key factor in our experiment.

The Morris water maze (Figure 4) demonstrates the degree to which the mice desire to find a way out of the swimming maze test, which assessed the ability of tottering mice to learn and/or remember as compared to the +/+ mice. Standardized behavior tests such as the Morris water maze are published, well-established tests that will further aid us in gaining an answer to the proposed research questions. It is assumed that mice, while very good at swimming, do not prefer to swim and will look for a way out of the water maze. They should be motivated to learn how to get out of the water and then remember what they have learned. However, if the mice have very low levels of anxiety this may result in lower motivation in learning how to get out of the water in the water maze. Such low motivation might then cause misinterpretation of the maze testing results. To illustrate, we discarded "floating" mice from our study. We defined floating mice as those mice that remained motionless in the water for more than 10 seconds. Crawley (1999) examined this floating behavior and discusses that it is a measure of depression-like behavior because the animal has "given up" on escaping.
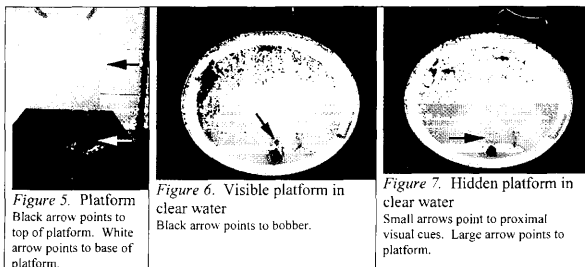
Animals

Male C57B/6J mice aged 2 to 6 months old were utilized for the behavioral tests. The animals were housed in clear plastic cages within a temperature-controlled room (21-23°C) with a 12 hour light-dark cycle. Three to five mice were housed in one cage. They had free access to food and water.

Figure 4. Morris water task setup
Arrow points to base of platform.
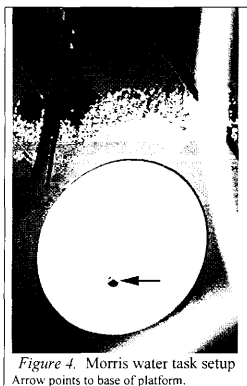
Morris Water Maze

Experiments were conducted in the light using a Morris water maze with close and distant visual cues to allow the mice to develop spatial learning. The Morris water maze consisted of a white plastic/polyurethane circular pool (74 cm in diameter, 38 cm in height) filled to a depth of 23 cm with $26\pm1°C$ tap water colored with powdered milk, which was changed every day, was located in a temperature-controlled room. Multi-colored shapes, camera tripod location (Fig. 4), cage placement, and other objects around and above the pool provided numerous distant visual cues. Additionally, visual cues (the letters N, S, W, E were 10 cm tall) were drawn on the inside wall of the tub 2.5 cm above the water to serve as proximal visual cues (Fig. 7). The total circular area of the pool was theoretically divided into 4 quadrants (NE, SE, SW and NW), which was later plotted on a 13" television to obtain tracings of mice swim paths. The mouse was required to find a (Figure 5, 6, 7) 8x8 cm clear plexiglass platform (height=21.5 cm) submerged 1.5 cm below the surface of the opaque water, which was present in the NE

Figure 5. Platform
Black arrow points to top of platform. White arrow points to base of platform.

Figure 6. Visible platform in clear water
Black arrow points to bobber.

Figure 7. Hidden platform in clear water
Small arrows point to proximal visual cues. Large arrow points to platform.

quadrant (between the center of the tub and the wall). Mice underwent 4 trials each day for a minimum of 3 training days with the platform located in the same position: NE quadrant (halfway between the center and the wall of the pool). The trial consisted of randomly placing a mouse into the water, facing the wall of the pool, in the SE, SW, or NW quadrants. Each trial lasted a maximum of 60 seconds in duration, and the mice were immediately removed from the platform in order to serve as positive reinforcement for finding the hidden platform. To achieve learning, criteria included: finding the platform in all four trials, climbing onto the platform in less than 30 seconds, and undergoing a minimum of 3 training days (maximum training length=10 days, after which the mice were tested in the probe trial).

Once the training days were completed and the mice reached criteria or had been trained a total of 10 consecutive days, they were videotaped in a "probe trial". This video trial kept all variables constant except the presence of the platform. For this trial, the platform was removed and the mice were videotaped for 60 seconds to record the swimming path, determine the mice's favored quadrant (percentage of time spent per quadrant), and calculate the number of crossings where the platform was located during the training days. Our expectation was that the +/+ mice would spend most of their time

in the NE quadrant (where the platform should be) and/or directly swim to the platform's location, and generate the most number of crossings.

In addition, we tested visual acuity by placing red and white bobbers on the typical Morris water maze platform (Fig. 6) so the mice would associate the visual cue (bobber) with the location of the platform. Since visual cues played a role in locating the platform, we decided to see if there was a significant difference between association, memory acquisition, and vision in −/− and tg/tg mice. We conducted the experiment similar to the hidden platform experiments; however, the only change was the addition of the red and white bobber to the platform for every training period.

RESULTS

While training both wildtype and tottering mice, we determined "achieved learning" by assessing each group of mice housed in the same cage. In pre-experimental training (for the maze operators), we discovered that tottering mice were not consistent with meeting "achieved learning" criteria. For example, some tottering mice achieved criteria on the third training day and appeared ready for the probe trial. However, when these same mice were trained an extra number of days (4-7), these animals were inconsistent with meeting the criteria for learning on the subsequent training days. Therefore, we decided to train all the mice in each cage as a group before the probe trial in order to gain an average performance time. Furthermore, for the animals that did not reach criteria, we continued training them for a maximum of 10 days. Day 10 of training was concluded as the maximum time for training because mice assessed after this time period clearly showed no acquisition of learning.

The wildtype mice, which lack calcium channel deficiencies, performed well in both visible and hidden platform tests (Figs. 8 and 9). In the typical Morris water maze (hidden platform task), the wildtype mice achieved learning in 3 training days and found the platform in an average of 10 seconds (Fig. 9). There was not a significant difference between the performance by the wildtype visible platform and wildtype hidden platform groups.

When comparing the tottering mice presented with a visible platform versus hidden platform, the results suggest that these mice are able to relate visual cues to locate the platform (Figs. 8 and 9). Previous literature by McNamara and Skelton (1993) revealed that spatial learning, not cue learning (visual association), was impaired by hippocampal damage. While the majority of the tottering hidden platform group required a minimum of 7 days of training, their average time to climb the platform was about 10 seconds greater than the tottering mice in the visible platform group (Fig. 9). This suggests that the visual cue located directly at the platform enhanced learning in these mutant mice.
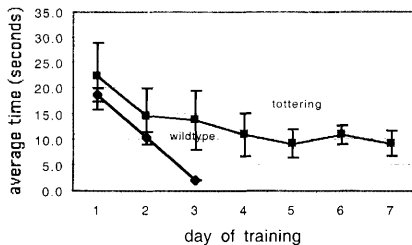
Figure 8. Visible Platform Training
Days of training for wildtype and tottering mice trained with the visible platform with standard error of the mean bars.
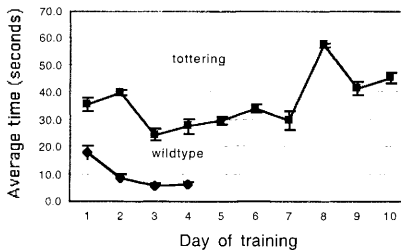

Figure 9. Hidden Platform Training
Days of training for wildtype and tottering mice trained with the hidden platform with standard error of the mean bars. Notice the increase in average time to find the platform for the tottering mice trained with the hidden platform.

There was a notable difference between the average finishing time of the tottering mice trained using the hidden platform (35 seconds and 7 training days) and wildtype mice (10 seconds and 3 training days) (Fig. 9). Our results provide evidence for the possible negative effects of the calcium ion channel deficiencies on learning and memory.

Secondly, when comparing wildtype and tottering mice with respect to the number of seconds it took to find the visible platform, the downward slope for the wildtype visible platform group was steeper, which depicts a decrease in time to find the platform over the number of training days (Fig. 8). This was also true for the hidden platform, but it was more pronounced with the visible platform. The steeper downward slope suggested that the wildtype mice may be acquiring learning and memory at a faster rate than the homozygous tottering mice.

From the video taped trials, we assessed the first ten seconds of movement in the maze because we expected the mice to immediately react to the stressful surrounding by initially locating and climbing the platform. We calculated the "number of direct swims (DS) and favored NE quadrant (NE)" in one category, while noting random swims (RS) in a separate category. Plotted on a bar graph, 62.5% of the +/+ mice and 30.77% of the tg/tg mice fit the DS/NE category. The low percentage of tg/tg mice in the DS/NE category also supported our hypothesis that these calcium channel-deficient mice have reduced spatial learning and memory (Fig. 10).

In order to properly document learning in wildtype and tottering mice, we counted the number of platform crossings (where the platform would be) in the probe trial as well as calculated the percentage of time the mouse spent in each quadrant. The wildtype mice trained with the hidden platform showed the greatest number of crossings over the region where the platform was located during training during the full 60 second probe trial (Fig. 11). The wildtype mice averaged 7.56 platform crossings with the hidden platform test, while the wildtype mice trained with the visible platform averaged 5.09 platform crossings (T-test, p=0.02).
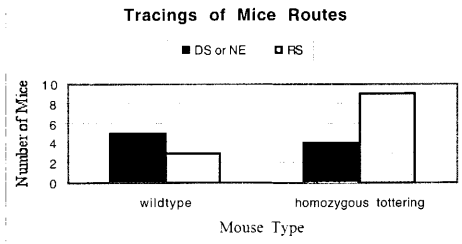
Tracings of Mice Routes

■ DS or NE   □ RS

Figure 10. Tracings of Mice Routes
The graph illustrates the greater number of +/+ mice that found the platform (DS/NE) as
compared to the tottering mice, which showed a greater number of random swims (RS).

In depicting the percent of time spent in a particular quadrant, the wildtype mice
trained with the visible platform showed a significant preference for the quadrant where
the platform was located during training. The wildtype mice trained with the hidden
platform performed similarly in that they also spent a much higher percentage of time in
the NE quadrant. This evidence further strengthens our hypothesis because the wildtype
mice do not have alpha-1A calcium ion channel subunit deficiencies. Therefore, they
were able to recall their memory from the training days to show that learning and
memory storage/retrieval had occurred.

Contrary to the wildtype mice performance, the tottering mice clearly portrayed a
lack of spatial learning and memory. Tottering mice trained with the visible platform
and tottering mice trained with the hidden platform demonstrated a decreased number of
crossings as compared to the wildtype mice trained with the visible platform (least
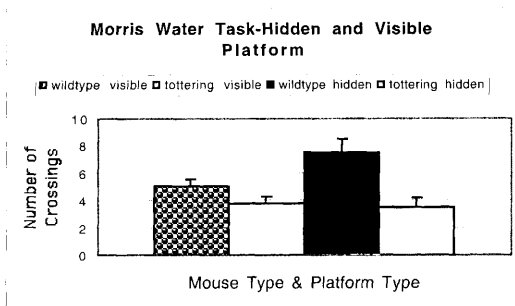number of crossings of the wildtype mice) (Fig. 11).

Morris Water Task-Hidden and Visible Platform

| ▨ wildtype visible ▢ tottering visible ■ wildtype hidden ▢ tottering hidden |

Figure 11. Number of Platform Crossings
Wildtype mice trained with the hidden platform showed the greatest number of platform crossings, followed by wildtype mice trained with the visible platform, tottering mice trained with the visible platform, and tottering mice trained with the hidden platform.

Another interesting result we found was the close similarity between the number of crossings for both groups of tottering mice. The tottering mice trained with the visible platform averaged 3.8 platform crossings for the probe trial, while the tottering mice trained with the hidden platform averaged 3.55 platform crossings (Fig. 11). This does not reflect the wider difference in number of platform crossings between the wildtype mice trained with the visible platform compared to the wildtype mice trained with the hidden platform.
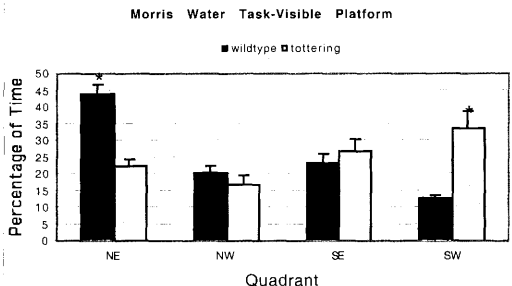
Morris Water Task-Visible Platform

■ wildtype □ tottering

Figure 12. Morris Water Task-Visible Platform

Wildtype mice showed the greatest amount of time spent in the NE quadrant, whereas the tottering mice spent most of their time swimming in the SW quadrant. Asterisks denote a significant difference (p<0.05 for the single factor ANOVA).

Contrary to the results obtained with the wildtype mice, neither group of tottering mice showed a clear preference for the NE quadrant during the probe trials (Fig. 12). Even with a proximal visual cue to facilitate association with the platform, the tottering mice continued to perform worse than the wildtype mice. However, the tottering mice trained with the visible platform did outperform the tottering mice trained with the hidden platform. This indicated that the tottering mice were capable of seeing and using the visual cues to help them locate the platform. Yet, this improvement did not reach the levels of either of the mouse groups. We expected the tottering mice trained with the visible platform to perform better than the tottering mice trained with the hidden platform.

The tottering mice trained with the hidden platform showed no significant difference in selecting the quadrant where the platform was located (NE) (Fig. 13). These mice only spent, on average, 21.8% of their time swimming in the NE quadrant.
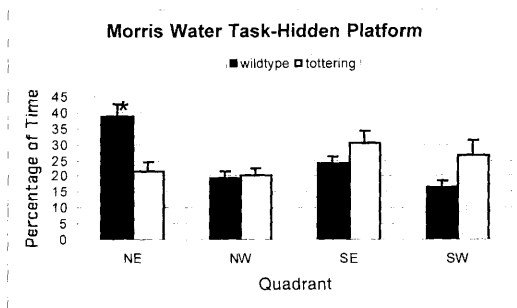
Figure 13. Morris Water Task-Hidden Platform
Wildtype mice showed the greatest amount of time spent in the NE quadrant, whereas the tottering mice spent most of their time swimming randomly throughout each quadrant. Asterisks denote a significant difference (p<0.05 for the single factor ANOVA).

<u>DISCUSSION</u>

Although difficult to establish a conclusive definition of learning, Dimond supports Champion's (1969) definition: "permanent changes in behaviour resulting from practice and experience." (Dimond, 1980). Learning allows information to be processed for storage and proceed to the major learning areas of the brain (Dimond, 1980). By analyzing previous literature from Bishop and Henry (1971), Dimond states that a neural basis of spatial navigation stems from a topographical projection of visual cues onto the visual cortex (Dimond, 1980). Dimond continues saying that the site associated with identification of objects in space is the visual cortex (Dimond, 1980). Finally, Dimond refers to Bishop and Henry's (1971) work in recognizing two different aspects of spatial perception: consistency and the pattern of perception (Dimond, 1980). For example, the relationship of the eyes to the brain to the hands and feet are regulated by the spatial perception of the individual (Dimond, 1980). Space, although recognized as homogeneous, is extensively mapped in a specialized topographical map (Dimond, 1980).

Before the probe trials were conducted, we thought the wildtype mice trained with the visible platform would perform better because they had the advantage of the visual cue to help locate the platform. However, the results seem to show that the wildtype visible platform group did not perform as well on the probe trials because the visual cue was removed. Therefore, they could have been less likely to use more distant visual cues to locate the platform. If the wildtype mice trained with the visible platform learned the location of the platform by utilizing the bobber marker, they may become lost or spatially disoriented since this visual cue was removed during the probe trial. Yet, if they achieved learning based upon the visual cues on the inside wall of the tub, then it seems they would have less difficulty in locating the platform (by crossing the place where the platform was normally located) for the probe trials.

On the contrary, the tottering mice trained with the visible platform and the tottering mice trained with the hidden platform did not demonstrate a difference in learning whether the bobber marker was present or not. This data is interesting because

the wildtype mice trained with the visible platform (5.09 average crossings) actually had a lower number of platform crossings than the wildtype mice trained with the hidden platform (7.56 average crossings). The tottering mice trained with the visible platform may have used the inner tub wall visual cues instead of relying only on the bobber marker. We currently have no clear explanation for this finding.

According to Fordyce and Wehner (1993), spatial learning is dependent upon hippocampal neural activity. The evidence presented here supports that a decrease in spatial learning in tottering mice could be associated with a lack of hippocampal function. The close similarity in number of crossings among the two separate groups of tottering mice is possibly due to their deficient alpha-1A calcium channel subunits, which could effect learning and memory in the hippocampus. Part of the limbic system serves as the major learning circuit of the brain (Dimond, 1980). This subcortical area is linked to the cortex by way of the hippocampal formation, which is responsible for coding, distributing, and retrieving memory (Dimond, 1980). Since both groups of tottering mice exhibit the behavior of the defective alpha-1A subunit protein, it seems logical that they would react similarly to a spatial navigation test because they will have decreased ability to store or recall information regardless if a proximal visual marker is provided.

Surprisingly, there was a significant difference in the tottering mice trained with the visible platform, in that the mice favored the SW quadrant (33.8% time spent). This was unexpected because we assumed the mice would randomly swim through each of the quadrants. Explanations for this occurrence stem from the possibility that: the mice feared the handler who stood to the East of the tub and the camera tripod positioned to the South of the tub; the tottering mice may have been confused as to the location of the platform when the marker was withdrawn in the probe trials; "floating" time that was less than 10 seconds may alter results since these mice were allowed to finish testing.

Interestingly, homozygous tottering mice seem to act similarly to wildtype mice that were injected with an NMDA antagonist, which seems to impair a particular region of the hippocampus. Hölscher's findings concluded that the mice injected with an NMDA antagonist showed signs of ataxia and impaired general sensorimotor coordination (Hölscher, 1999). Additionally, both groups (tottering mice and wildtype mice injected with a NMDA antagonist) seem to respond in the same manner when

placed in the Morris water maze: once the animal bumped into the platform, it made no attempt to climb onto the platform for the first several training days (Hölscher, 1999).

Overall, there is good supporting evidence that defective alpha-1A calcium channel subunits and other molecules or agents that hinder calcium entry into neurons, play a significant role in decreasing learning and memory in mice.

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EDUCATION   **Texas A&M University**, College Station, Texas
Bachelor of Science in Biomedical Science
Anticipated May 2001
Cooper High School, Abilene, Texas
May 1997

RELEVANT COURSE WORK

Honors Biology I & II	Chemistry I and II	Organic Chemistry I and II
Honors Neurology & Neuroscience	Physiology I and II	Biochemistry I and II
Honors Histology	Immunology	Microbiology
Pharmacology	Food Toxicology & Safety	Intro to Diseases

HONORS AND AWARDS

1st place undergraduate poster presentation (Veterinary Medicine, Student Research Week), Sigma Xi (Associate Member), President's Student Service Challenge Gold Award, The National Dean's List, University Undergraduate Research Fellows Program, Phi Kappa Phi (national honor society, top 10% juniors and seniors), National Society of Collegiate Scholars, Golden Key National Honor Society, Alpha Epsilon Delta (honors premedical society), Phi Eta Sigma (freshman national honor society), Dean's Honor Roll, Distinguished Student List, Texas A&M University Honors Program, Texas A&M University's Academic Incentive Scholarship, Kiwanis Club Scholarship, Abilene Business Women's Association Scholarship, Taylor Jones Haskell Counties Medical Alliance Scholarship

EXTRACURRICULAR ACTIVITIES

Golden Key National Honor Society, Phi Eta Sigma Social Committee Chairperson, Alpha Epsilon Delta, Premedical Society, Biomedical Science Association, Habitat for Humanity (helped plan annual Fun Run), Big Event, Replant, Texas A&M Club volleyball team, intramural volleyball, taekwondo (1st degree black belt, student instructor), American Medical Student Association, CPR certification (health care provider for adult, child, and infant), Health For All ClinicVolunteer, Summer Premedical Enrichment Program (University of Cincinnati College of Medicine), Camp John Marc Counselor, work-study at Institute of Developmental & Molecular Biology, student tutor (MATH131, CHEM101, CHEM102), St. Joseph Regional Health Center (ER Volunteer), Columbia Medical Center (ICU Volunteer), Shadow Program (Hendrick Health System), National Youth Leadership Forum on Medicine (Boston, MA)

INTERESTS

Violin, Piano, Roller Hockey

REFERENCES

Dr. Louise C. Abbott
Dr. Max Amoss
Dr. Newell McArthur